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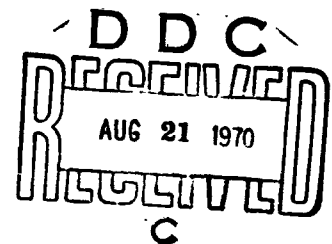
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PAPER NO. 2

## THE EFFECT OF CARBON MONOXIDE ON HUMAN PERFORMANCE

Peter J. Mikulka, Captain, USAF  
Robert D. O'Donnell, Captain, USAF  
Patrick E. Heinig, Lieutenant, USAF  
and  
James Theodore, Major, USAF, MC

Aerospace Medical Research Laboratory  
Wright-Patterson Air Force Base, Ohio



### INTRODUCTION

Carbon monoxide (CO) has become an important byproduct of increasing industrialization. The potential risk of this toxic substance applies not only to the general population, but also to specific subpopulations (i.e., respiratory and cardiovascular patients, military, etc.). This risk is considered extremely important in the aerospace environment where human performance is carried to its extreme limits in high performance aircraft and space systems. Any effect of low levels of CO on performance must be considered serious insofar as it affects the system operator. Mechanisms of CO action must be correlated with performance decrements in order to arrive at realistic guidelines on atmospheric control for man.

The central nervous system is extremely sensitive to oxygen deprivation and is considered the primary locus of CO induced effects, since the major mechanism of CO action is mediated through tissue hypoxia (Dinman, 1968). It is generally believed that subjective symptoms rarely occur below carboxyhemoglobin (COHb) levels of 20 percent, while most acute signs of cardiovascular, respiratory, and central nervous system embarrassment occur at COHb levels greater than 30 percent (Haldane, 1927). However, a number of investigators have indicated that the central nervous system is impaired at COHb levels as low as two to five percent. MacFarland, Roughton, Halperin, and Niven (1944) demonstrated impairment of visual discrimination with COHb levels of four percent. Lillenthal and Fugitt (1946) reported impaired flicker fusion at an altitude of 6000 feet with COHb levels of five and ten percent. Decrements in limb coordination have been shown with the same COHb levels at ground level atmospheric pressure (Trouton & Eysenck, 1961). Consistent impairment in cognitive and psychomotor performance has been noted by Schulte (1963) at five percent COHb, and some tendencies for disruption as low as two percent COHb. Finally, Beard and Wertheim (1967) have shown decreased auditory discriminability of tone lengths with COHb levels of approximately four to five percent.

However, not all investigators have shown such effects at low levels of COHb. Clayton, Cook, and Frederick (1960) found no association between COHb level and automobile accidents. Dorcus and Weigand (1929) found no decrements in cognitive and psychomotor tasks with COHb levels as high as 25 to 35 percent. Vollmer, King, Birren, and Fisher (1946) found no changes in flicker fusion, visual perimeter, or ataxia as a function of COHb levels up to 22 percent. However, their study was run at simulated 10,000 and 15,000 foot altitudes, and the hypoxic contribution of those altitudes may have masked possible CO effects.

Cigarette smokers are estimated to have a constant COHb level between 3.8 and 6.8 percent. Further, 12 to 14 percent of employed individuals have occupations in which there is a likelihood of exposure to high levels of CO (Goldsmith and Landaw, 1968). Given these facts, plus the studies which suggest decremental effects under COHb levels that the average smoker carries within his system, one could conclude that a large proportion of our population is operating at a depressed level of performance. It therefore becomes important to determine what effects are present, their mechanism of action on the organism, and the importance of these effects on specific performances (i.e., driving an automobile, piloting an aircraft, etc.).

Obviously these questions cannot be answered until a better understanding and correlation of the physiologic, biochemical and psychologic processes of the brain are known. At the present level of sophistication, Dinman (1968) contends that cerebral function should not be impaired at COHb levels of five percent or lower. If this assumption is true, then those data which show decrements with COHb levels of three to five percent must depend on some complex interaction or summation of elements which have yet to be defined.

The present study was undertaken to determine the effects of CO on relatively simple applied performance tasks. Analyses were planned which would reveal whether: (1) absolute performance levels changed as a function of CO exposure, and if (2) CO exposure changed the pattern of performance.

## METHOD

### Subjects:

The subjects (Ss) used in this study were 10 male university students between the ages of 19 and 22 years. All Ss were examined and medically certified to be in good health. All Ss were supposed to be non-smokers. However, after completion of this study, examination of the blood COHb levels indicated that one S was probably a smoker. He confirmed this when questioned and his data were excluded from all analyses.

### Environment:

All CO and control exposures were carried out in the Thomas Domes of the Toxic Hazards Division, Wright-Patterson Air Force Base, Ohio. The domes are a completely enclosed environmental system into which a given contaminant can be introduced and maintained at a given level. Air flow is controlled by a series of blowers and vacuum pumps, which produce a flow of 40 ft<sup>3</sup>/min, yielding a complete atmospheric change every 20 minutes. Each dome is roughly circular with a 12 foot diameter. Ss inside the dome can see into the surrounding room, but for the present study about eight feet of the dome windows were covered in order to eliminate distracting background movement from the S's field of view. However, it was considered important that during "rest periods" the Ss could see outside in order to preclude sensory restriction effect which could mimic or confound CO effects. Temperature was controlled between 68 and 74 degrees F, and dome pressure was held at 680 mm Hg during the experimental runs. Entrance to the domes was accomplished through an airlock, which allowed the interior environment to be kept stable.

### Experimental Measures:

Time Estimation: During each testing interval a series of estimates of a 10-second "empty" interval were made by each S. The S was asked to estimate 10 seconds, beginning on a signal from the experimenter (E). At the end of this estimated interval the S tapped an electronic switch and immediately began estimating another 10-second interval, etc. The E stopped this sequence when the S's last estimate exceeded the three minute test period. All the S's estimates were automatically recorded.

Critical Instability Tracking Task (CITT): The CITT requires the S to stabilize a statically unstable controlled element by closing a compensatory loop around the system (Jex, 1967). Essentially, the S is required to keep a needle on a display dial from going off scale by manipulating a control stick. Referring to figure 1 it can be seen that any output from the integrator will be fed back through the summing amplifier in such a way as to cause the output of the integrator to increase. If the S, by moving his control stick, generates input to the summing amplifier which exactly cancels the input fed back from the integrator, the output (needle on dial face) will remain stationary. Any slight error in timing or the amplitude of the response will cause the output to change. The S's optimal strategy is to keep his control stick displacement exactly proportional to the system output (needle deflection). For the purpose of this experiment the gain control was set to increase linearly over time. In essence, this multiplies the output from the summing amplifier returning to the integrator, causing increased input to the needle, which requires the S to make greater and quicker compensations. As the gain increases a point will be reached where the S cannot possibly respond quickly and accurately enough to "maintain control". This point of control loss is converted into a difficulty level score, and serves as the basis for evaluation of the S's tracking ability. The CITT has been analyzed on theoretical grounds to be sensitive to a number of stressors including hypoxia, drugs, g-levels, low temperature, and secondary workloads (Rosenberg and Jex, 1966; Jex, 1967); and on the basis of the

describing functions involved appears to be most closely related to performances requiring a great deal of perceptual-motor coordination and high requirements for speed and accuracy.

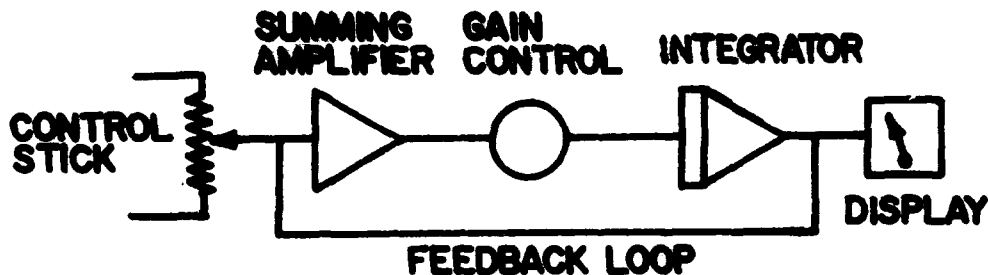


Figure 1. DIAGRAMMATIC SCHEME FOR CRITICAL INSTABILITY TRACKING TASK (CITT).

**Pensacola Ataxia Battery:** Since one of the effects of relatively severe CO exposure is the loss of balance and other vestibular symptoms, the Pensacola Ataxia Battery was used. This battery consists of a number of balancing tasks performed either on narrow rails or on the floor (Greybell and Fregley, 1965). For the present experiment the following tasks were used:

1. The Sharpened Rhomberg task (SR) in which the S attempts to stand for 60 seconds in a heel-to-toe position with his arms folded and eyes closed.
2. Walk Eyes Open test (WE/O) in which the S attempts to walk a 3/4 inch wide rail with his arms folded and eyes open.
3. Stand Eyes Open test (SE/O) in which the S attempts to stand on the above rail for 30 seconds with his arms folded and eyes open, and with his feet in a heel-to-toe position.
4. Stand Eyes Closed test (SE/C) in which the S attempts to stand for 60 seconds on a 2-1/4 inch wide rail in a heel-to-toe position, arms folded, and eyes closed.
5. Stand on One Leg Eyes Closed test (SOLEC) in which the S tries to stand on one leg, eyes closed, and arms folded for 30 seconds. This is done alternately on both legs resulting in SOLEC-Right and SOLEC-Left.
6. Walk On Line Eyes Closed test (WOLEC) in which the S attempts to walk a 12 foot line with his arms folded and eyes closed in a heel-to-toe manner.

Design and Testing Procedure:

Each S spent three sessions in the Thomas Domes at 0, 50, and 125 ppm CO. The order of exposures was counterbalanced to avoid possible sequence effects. With the removal of the smoker and due to an error on the part of the technician controlling the exposure contaminant, which resulted in one S being run a fourth time, the resulting order was: three Ss with a A-B-C presentation order (0-50-125 ppm), three Ss with C-A-B, two Ss with B-C-A, and one with B-A-C.

A double-blind procedure was used throughout this experiment. At no time did the Es know what was the atmospheric composition in the dome. Also, all immediately involved support technicians were included in the double-blind coverage.

Ss were scheduled at either 8 AM or 1 PM depending on their availability. Exposure time began at the moment the S entered the dome. Headphones were given the S through which he was in continuous touch with the Es. After a final check of all recording and communication equipment the E left the dome through the airlock. The S was then instructed to relax for a few minutes. The first series of measurements was taken after the S had been in the dome exactly 15 minutes.

A performance session consisted of five trials on the CITT, three minutes of time estimation, and then five more tracking trials. This sequence usually ran from 13 to 16 minutes. After completion of the session the S was instructed to relax for about 15 minutes until the next testing session.

Each dome exposure lasted three hours. The Ss were tested 15 minutes out of each half hour, resulting in the following test intervals after dome entrance: 15-30 min, 45-60 min, 75-90 min, 105-120 min, 135-150 min, and 165-180 min. After 90 minutes of exposure the E entered the dome allowing the S to walk around and stretch. This was done to reduce the possibility of fatigue and boredom from being in a constant position for three hours.

At the completion of each run the S was immediately removed and 10 ml of blood was taken for hematocrit, hemoglobin, and COHb determinations. Then, the S was taken to an adjoining room and given the Pensacola Ataxia battery. Following this the S was asked to breathe 100 percent oxygen for ten minutes.

Following completion of the main study five Ss were exposed to 200 ppm CO and three Ss to 250 ppm. The procedures were basically the same with the following exceptions: (1) the double-blind was no longer in effect since these runs were made for exploratory purposes, (2) counterbalancing was no longer possible, and (3) the Ss were specifically told that they would be receiving higher levels of CO. Because of the lack of counterbalancing and the reduced sample size no analyses were performed on these data.

## RESULTS

Carbon Monoxide Exposure Levels:

Carboxyhemoglobin (COHb) determinations were made on the venous blood for each S after every experimental session. These determinations were done by a modified gas chromatographic method of Dominguez, Christensen, Goldbaum, and Stembridge (1959). The results of the COHb analyses are presented in table I. It can be seen that the COHb levels reflect a direct relationship with the level of ambient CO. These data are in remarkable agreement with the CO uptake curves based on time, exposure concentration, and rates of ventilation constructed by Forbes, Sargent, and Roughton (1945). The exception to this was S #10 who had a COHb level of 3.5 percent in the 0 ppm condition. This S later admitted he was a smoker. His level of 3.5 percent COHb falls into the range of 2.3-3.8 percent COHb reported for light smokers (Ringold, Goldsmith, Helwig, Finn, and Schuette, 1962). Hematocrit levels for all Ss ranged from 42 to 52%, and hemoglobin levels ranged from 12.8 to 17.6 gms percent with no significant changes due to CO exposure.

TABLE I  
CARBOXYHEMOGLOBIN LEVELS FOR ALL SUBJECTS AT EACH EXPOSURE  
(PERCENT)

SUBJECT	0 PPM	50 PPM	125 PPM	200 PPM	250 PPM
1	0.7	2.8	6.5		
2	1.2	2.4	6.6		
3	0.8	2.7	6.5	9.8	
4	1.0	3.1	7.4		13.1
5	1.0	3.0	6.8		
6	1.3	3.6	6.8	10.1	11.9
7	1.1	3.3	6.4	10.0	
8	0.9	3.0	6.6	10.9	
9	0.6	2.9	6.2	10.9	12.1
10	3.5	4.5	7.4		
MEAN*	0.96	2.98	6.64	10.35	12.37

\* does not include values for S #10

Subjective Reports:

All Ss were interviewed informally after each run to determine the incidence of subjectively perceived symptoms. In the 39 experimental sessions carried out, only one S reported any symptoms. This occurred in S #4 during exposure to 250 ppm, who reported a "slight" headache which he said did not interfere with his performance. Further, none of the Ss displayed an ability to indicate whether or not CO had been present during a given experimental run.

Critical Instability Tracking:

The mean difficulty level was obtained for each set of ten trials for every test interval. Therefore, each S had six scores for each exposure level and these were the basis for the analyses.

Since the uptake of CO within the body is a cumulative process over time, it is important to compare not only the differences among the conditions at each test interval, but also to evaluate the trends occurring over the three hours of exposure. Grouped data representing the mean scores for nine Ss at each test interval are presented in figure 2. Initially, it can be seen that no trend toward poorer performance appeared in any of the conditions. In fact, there was a general tendency for all conditions to improve over time. Therefore, each condition was tested independently to determine if performance changes had occurred. These analyses yielded F ratios of 1.84 (for 0 ppm), 1.81 (for 50 ppm), and 2.23 (for 125 ppm), with  $P = >.10$ ,  $>.10$ , and  $<.10$ , respectively. Thus, when considered separately no condition showed a clearly reliable change in tracking performance over time, and the one condition that showed any change at all indicated that tracking performance became slightly better during exposure to 125 ppm. Reference to figure 3 shows that the Ss tracking under 200 ppm and 250 ppm did not differ from their control runs under 0 ppm. In absolute terms, these findings answer one of the primary concerns of this experiment: No decrement in tracking performance was found as a result of exposure to CO levels up to 125 ppm, and quite possibly as high as 250 ppm.

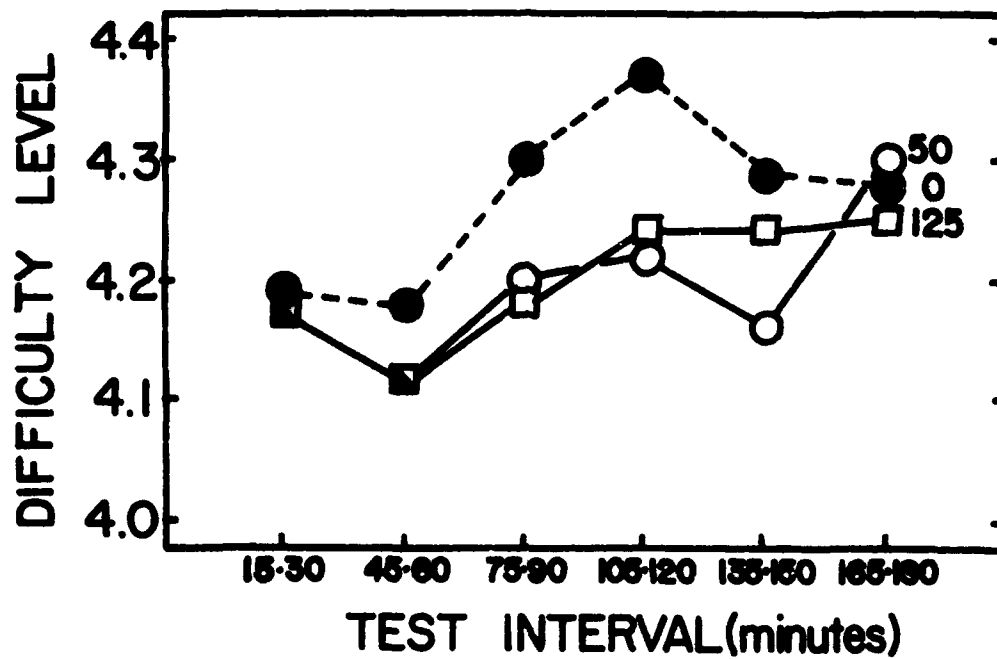


Figure 2. MEAN SCORES FOR CRITICAL INSTABILITY TRACKING TEST (9 S<sub>8</sub>)



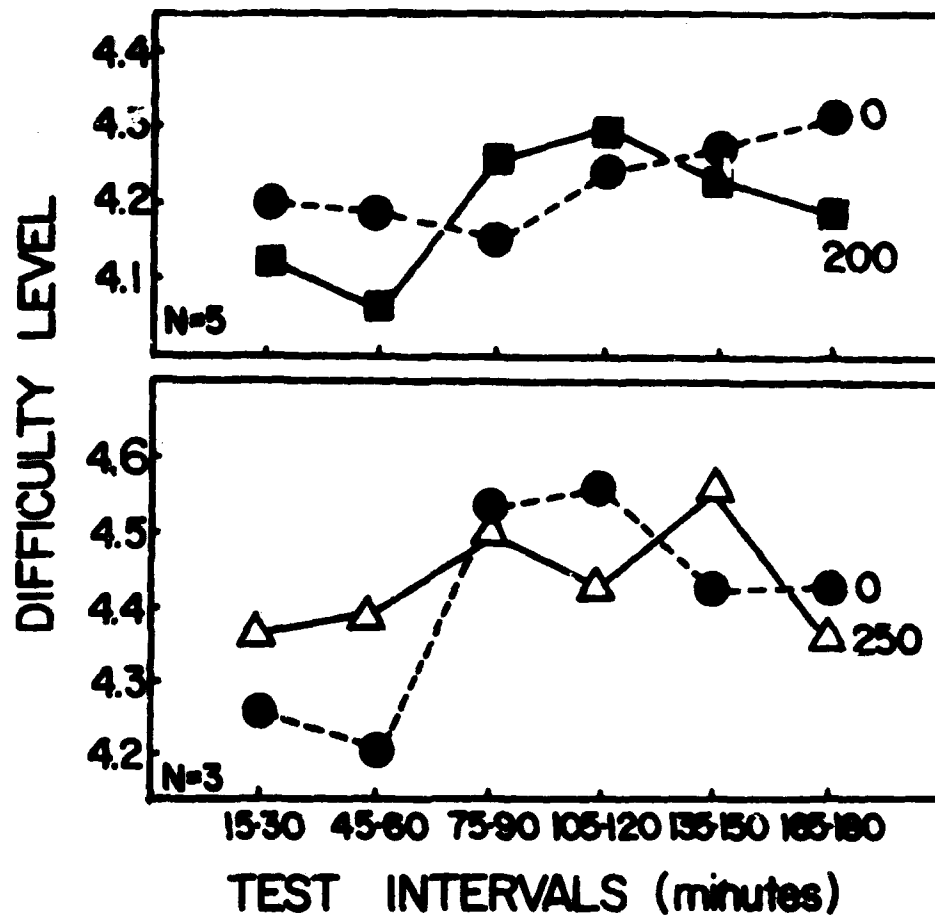


Figure 3. CRITICAL INSTABILITY TRACKING TEST (8 Ss)

In order to determine if there was a relative difference between scores with and without CO present, these data were subjected to repeated-measures analyses of variance at each test period. The results of these analyses are presented in table II. It can be seen from these analyses that there was a significant difference in performance levels of these three conditions at the 105-120 minute interval, which decreased by the 135-150 interval and completely disappeared by the last interval. Newman-Keuls analyses (Winer, 1962) were performed on the 105-120 and 135-150 intervals. These indicated that performance under 0 ppm was better than either of the CO conditions at the former interval ( $P < .05$ ), while performance under 0 ppm was only marginally better than the 50 ppm condition, though not different from 125 ppm at the latter interval. At both intervals the two CO levels did not perform significantly different from each other. These results suggest that CO exposure resulted in a relative performance change compared to the control condition. However, this effect was transitory reaching a maximum at 105-120 minutes and diminishing as duration of exposure increased. Further, examination of the performance trends shows that these effects were not due to any decrement in the absolute level of performance under CO, but the result of a transitory increase in performance under 0 ppm (see figure 2). Trend analyses were done on each condition over the six test periods (Winer, 1962). The results are presented in table III. These analyses indicate that a large proportion of the 0 ppm trend was composed of linear and quadratic components, at 50 ppm the linear and quartic components composed the largest proportion of the curve, and at 125 ppm the linear component accounted for most of the trend with a smaller cubic component. Examination of figure 2 shows that there are distinct differences in the shapes of the curves and that these are most notably evident in a stronger tendency for linearity over time in the 125 ppm condition and in a much larger quadratic or singly-humped performance over time in the control condition than in any other. Also, average performance at 50 ppm shows a greater tendency to be erratic as evidenced by the large quartic component.

Inspection of the individual performance curves revealed no consistent differences as a function of CO exposure. In four of the nine Ss tracking was generally better under 0 ppm than in the other conditions. In only one S was tracking under 125 ppm consistently the poorest, while four Ss performed worst under 50 ppm.

As CO uptake increased over time, performance would be expected to show a decrement if there were any simple relationship between the two. However, in no S was an overall time-related decrement seen in any of the CO runs up to and including 250 ppm. In fact, several Ss show a remarkably consistent improvement in tracking as a function of time.

TABLE II  
ANALYSES OF VARIANCE ON TRACKING SCORES FOR EACH TEST INTERVAL

INTERVAL	SOURCE	df	MEAN SQUARE	F RATIO
15-30	Between $S_s$	8	.220	.03
	Within $S_s$	18	.020	
	CO	2	.001	
	Residual	16	.023	
45-60	Between $S_s$	8	.218	.41
	Within $S_s$	18	.025	
	CO	2	.011	
	Residual	16	.027	
75-90	Between $S_s$	8	.389	2.35
	Within $S_s$	18	.017	
	CO	2	.035	
	Residual	16	.015	
105-120	Between $S_s$	8	.358	4.53**
	Within $S_s$	18	.018	
	CO	2	.058	
	Residual	16	.013	
135-150	Between $S_s$	8	.410	2.86*
	Within $S_s$	18	.016	
	CO	2	.039	
	Residual	16	.014	
165-180	Between $S_s$	8	.412	.66
	Within $S_s$	18	.009	
	CO	2	.006	
	Residual	16	.009	

\* significant at .10 level

\*\* significant at .05 level

TABLE III  
PERCENT OF GIVEN POLYNOMIAL COMPONENT PRESENT IN TREND ANALYSIS

COMPONENT	0 PPM	50 PPM	125 PPM
Linear	37.9	45.0	62.4
Quadratic	31.3	1.0	0.9
Cubic	9.4	0.9	24.7
Quartic	5.8	41.6	11.6

#### Time Estimation:

Under the procedure for time estimation in this study each  $S$  made a series of estimates during each test interval. The mean of these individual estimates was taken for each interval and these means constituted the raw data for subsequent analyses.

The mean performance curves for time estimation under the three experimental conditions are presented in figure 4. It is evident that although there is some separation between the different conditions, no overall trend to over- or under-estimation occurred as a function of CO uptake. There is a slight tendency for all conditions to increase accuracy over time. A very similar pattern can be seen for the  $S$ s exposed to 200 ppm and 250 ppm (figure 5). Under all conditions time estimation remains remarkably consistent.

In order to test for relative differences among the 0 ppm, 50 ppm, and the 125 ppm conditions, separate repeated-measures-analyses of variance were performed. The results are presented in table IV. The only significant difference occurred at the 135-150 minute interval, where time estimates under 50 ppm were longer than under the control condition, as determined by the Newman-Keuls test. Inspection of figure 4 reveals that this difference resulted from a decrease in the average estimates in the 0 ppm condition, and not from any change (away from the real 10-second interval) by the  $S$ s while under CO. The difference dealt with here is less than one second, and it is difficult to construe this as a significant distortion due to CO exposure, especially when this effect was not seen in the 125 ppm condition. At no point were the estimates under 125 ppm different from those under the control condition.

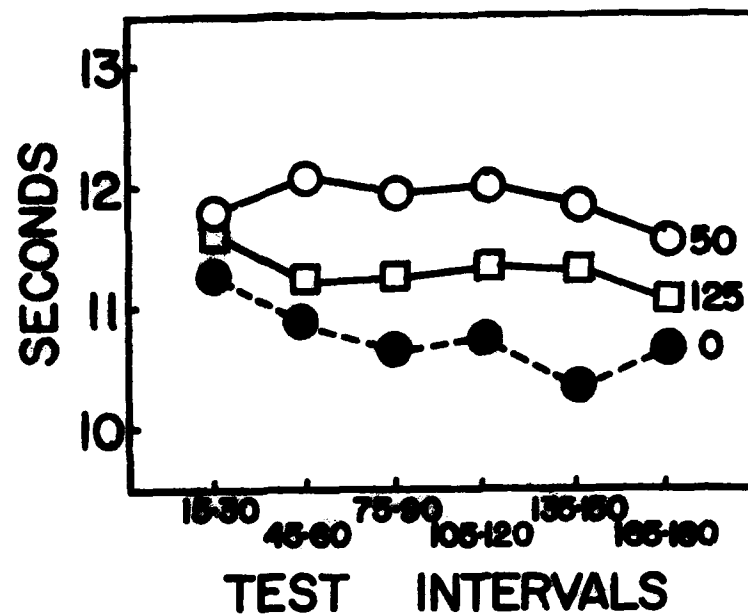


Figure 4. TIME ESTIMATION PERFORMANCE CURVES (0, 50, 125 ppm CO)

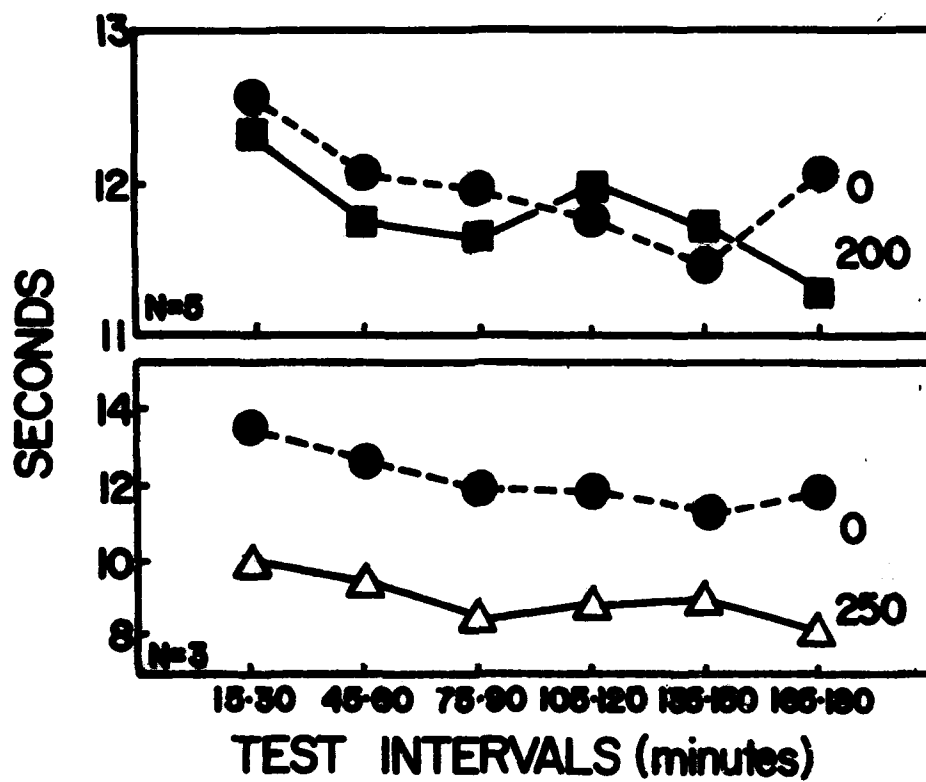


Figure 5. TIME ESTIMATION PERFORMANCE CURVES (200 and 250 ppm CO)

TABLE IV  
ANALYSES OF VARIANCE ON TIME ESTIMATION AT EACH TEST INTERVAL

INTERVAL	SOURCE	df	MEAN SQUARE	F RATIO
15-30	Between Ss	8	14.795	0.121
	Within Ss	18	3.718	
	CO	2	0.500	
	Residual	16	4.120	
45-60	Between Ss	8	13.109	0.824
	Within Ss	18	3.678	
	CO	2	3.090	
	Residual	16	3.751	
75-90	Between Ss	8	15.806	1.417
	Within Ss	18	2.678	
	CO	2	3.605	
	Residual	16	2.562	
105-120	Between Ss	8	18.201	1.663
	Within Ss	18	2.230	
	CO	2	3.455	
	Residual	16		
135-150	Between Ss	8	21.069	3.987*
	Within Ss	18	1.668	
	CO	2	4.980	
	Residual	16	1.249	
165-180	Between Ss	8	27.064	1.468
	Within Ss	18	1.233	
	CO	2	4.989	
	Residual	16	1.172	

\* significant at .05 level.

Inspection of the individual  $\bar{S}$  estimates similarly revealed no consistent effect attributable to CO. In five  $\bar{S}$ s time estimates were longer under CO conditions than under 0 ppm. However, for the other  $\bar{S}$ s one or both of the CO conditions gave more accurate estimates than the control condition. In two cases 125 ppm produced the longest estimates, while in five  $\bar{S}$ s the 50 ppm condition produced the longest estimates.

Further support for the lack of any CO effect on time estimation can be assumed from the performance curves of those  $\bar{S}$ s that received additional runs at 200 ppm and 250 ppm (figure 5). From these curves it is obvious that (1) there is a general tendency for estimates to improve over time, irrespective of contaminant level, (2) performance for the 200 ppm condition closely parallels that for its control run, and (3) under 250 ppm the  $\bar{S}$ s are more "accurate" in their estimates than under the 0 ppm condition, but more importantly there is no indication of a differential effect of CO over time.

In summary, it may be concluded that under the conditions of this study, no effect of CO levels up to 125 ppm and possibly as high as 250 ppm could be discerned on time estimation. In all conditions the estimated 10-second interval was longer than the real 10-second interval.

#### Pensacola Ataxia Battery:

The scoring on the Ataxia battery was accomplished using procedures recommended by Greybell and Fregley (1965).

The means for each of the tests are shown in table V along with the F ratios from the repeated-measures-analyses of variance. All the resultant F ratios were non-significant. Examination of the means for the respective ataxia test shows that there are no major differences within the means for a given test. In absolute terms, four tests (SR, SOLEC-L, SOLEC-R, & WOLEC) yielded better performance under the CO conditions than under 0 ppm. In only one case (SE/O) were the scores under the CO conditions worse than the control condition.

In view of these results, it is clear that CO exposure had no effect on the kinds of abilities measured by these tests.



TABLE V  
MEANS AND F RATIOS FOR ALL PENSACOLA ATAXIA TESTS

TEST	0 PPM	50 PPM	125 PPM	F RATIO
SR	204.00	221.00	217.00	0.758
WE/O	14.00	14.13	13.75	0.117
SE/O	32.25	29.75	30.88	9.090
SE/C	81.13	80.75	81.13	0.001
SOLEC-L	133.13	138.50	135.13	0.103
SOLEC-R	140.50	150.00	141.50	1.681
WOLEC	17.13	13.38	10.50	0.963

#### DISCUSSION

This study attempted to answer the question: does CO affect performance? In order to explore this question, a range of performance measures were taken: 1) time estimation of an "empty" 10-second interval, 2) tracking, and 3) ataxia. On an ordered scale these measures run from the heavily cognitive task of estimating time in which the S supplies his own counting stimuli, to the tracking task which requires the coordination of visual input with rapid motor responses, to a psychomotor task involving vestibular and gross motor controls for dynamic equilibrium. The results of this experiment indicated that three hours of exposure to CO levels up through 125 ppm produced no decremental effect in functioning and no consistently reliable pattern changes from the control conditions. Also, there is an indication that carrying the CO levels up to 200-250 ppm will produce no observable effect on the present battery of performance tasks.

The results of this study conflict with several other studies which have found performance decrements under low levels of CO. Beard and Wertheim (1967) using a temporal auditory discriminability task (perceptual) found major disruptions under 50 ppm CO. Unfortunately, they were unable to obtain accurate COHb determination, but given their exposure times it is possible to assume that their Ss did not have COHb levels above 5 percent. It should be noted that their Ss were confined in a soundproof audiometer booth with a total volume of 110 cubic feet. The Ss had no outside visibility and the tasks did not involve a great deal of kinesthetic, proprioceptive, or visual input to the Ss. In view of the fact that even moderate degrees of sensory or motor restriction can cause significant perceptual and motor distortions (Schultz, 1965), it is possible that any CO effects reported could be accounted for, or at least confounded, by sensory restriction effects. In contrast, Ss in the present study were confined in a large dome with an approximate free volume of 600 cubic feet. At all times the Ss could see out-

side the dome. At the midpoint of each run the Ss were allowed to get up and move around inside the dome. Additionally, the tasks involved a good deal of visual input and a significant amount of motor output from the Ss. These procedures were specifically intended to minimize any possible effects of sensory restriction or boredom, in order to yield a less contaminated estimate of CC effects. In view of this it may not be surprising that the present results do not show the perceptual errors seen by Beard and Wertheim (1967).

Schulte (1963) using a battery of psychomotor-perceptual tasks found performance disruptions with reported COHb levels comparable to those obtained after three hours of exposure to 125 ppm CO in the present study. However, there is reason to believe that Schulte underestimated the levels of COHb in his Ss, since he reports obtaining mean COHb determinations of 0.00 percent from Ss who were predominantly smokers, working in a large metropolitan area as firemen. Recent data indicate that 1.2 percent COHb is found in the average metropolitan non-smoker (which agrees well with the 0.96 determination in our non-smokers under 0 ppm CO), while the range for smokers in a metropolitan area runs from 2.3 to 6.8 (light smokers, noninhalers to heavy smokers, inhalers). Further, his findings of perceptual-cognitive decrements conflict with a study which failed to find any decrements of similar tasks with up to 25-35 percent COHb (Dorcus and Weigand, 1929). Also, examination of some of the reported decrements in cortically mediated tasks, such as arithmetic errors shows that the absolute number of errors at 1 percent COHb was higher than at all other levels up to 20 percent, except at two levels.

Trouton and Eysenck (1961) reported impairment in control precision and multiple limb coordination when COHb levels exceeded 5 percent, whereas Schulte (1963) found no change in muscular coordination as measured by reaction time, static steadiness, muscle persistence, and cranial reflexes. Consistent with this the present study failed to find any evidence for disruption in tracking or ataxia with average COHb levels of 6.6 percent. Several other studies failed to find disruption in performances which required a great deal of motor coordination until high levels of COHb were reached (Clayton, Cook, and Frederick, 1960; Forbes, Dill, DeSilva, and Van Deventer, 1937; Rockwell and Ray, 1967; Vollmer, King, Birren, and Fisher, 1946).

In summary, it can be said that the case for performance changes under low levels of carbon monoxide is not very compelling, and must await further experimental support. However, if there is no effect at these low levels of CO on performance, then we have a finding of significant practical importance.

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## DISCUSSION

DR. MACFARLAND (YORK UNIVERSITY OF CANADA): I know nothing about the background of this work other than what you told us today, but as a toxicologist who's done a great many experiments over 25 years or so, as I look at your graphs there's a suggestion that comes to me--it may be valueless here, but I'll throw it out for what it's worth. In looking at the various graphs you have presented of your results, it seems to me, superficially, two kinds of things. In some cases your curves are sort of intertwined and it is perfectly evident there's no significant difference in the CO groups versus the zero group. In some of the other graphs, however, you notice that the curves parallel each other but they are separated by a distance. This usually suggests to me, in the kind of work that I do, that there is probably some kind of systematic error that I've got in my studies, but--again, I throw this out as probably not true--it might be a difference in your analysis for carbon monoxide, or something wrong with the analytical instrument that throws it off by a certain increment. As I say, I don't suppose this is the case here, but the suggestion that comes out of this is that in some of the cases where the curves are separated, more or less parallel, --a good guess would be that there may be systematic error operating that you haven't identified.

DR. MIKULKA: On your point there, there are two things. Statistically, if you see differences in graphs that aren't held up, you really have no basis for saying anything. That's one thing. Secondly, one of the tasks, where you see the big differences in time estimation, was done because somebody else had used it and shown effects. Actually, the study was done with the assumption that there are effects there and we want to see just how big they are and what direction they are. Well, it turns out that the task of time estimation, although you think that everybody has an estimation of what ten seconds is, is a learning phenomenon that, as a function of exposure these guys got better, and even though the design was counterbalanced, a couple of subjects really showed improvement and happened to occur in the "wrong" groups, the CO exposed groups. It only takes one or two subjects who are consistently better to pull that whole thing off. You don't see it as markedly or at all in tracking performance. Everybody is trained very well in specific skills like piloting an aircraft, very complex motor tasks, where they trained maybe for 30 hours apiece. I don't know if that answers your question.

MAJOR THEODORE: I would like to answer the second part of his question about the question of analytic error. Roughton, who probably knows more about carbon monoxide uptakes in hemoglobin than anyone else in the world, constructed curves a number of years ago with Forbes as a senior author, and on his curves he took into account the rate of carbon monoxide passage through the lung, rate of uptake with hemoglobin.

By comparing over the time interval, since carbon monoxide uptake is related to time, the affinity of hemoglobin and rate of ventilation, he constructed these curves, and our data hit his curves on the point almost consistently to the point of disbelief. And the spread of our data was so small that as far as analytical error being part of this, I really doubt this, and theoretically it followed the prediction curves put forth by Roughton.

DR. BACK (Aerospace Medical Research Laboratory): Did you put anything on the board showing carboxyhemoglobin levels?

MAJOR THEODORE: No. I can give them to you; I know them.

DR. BACK: Maybe the audience would be interested in what they were for comparative purposes with Beard's work and others.

MAJOR THEODORE: Well, quickly, Goldsmith and Landau found 1.2% in the average metropolitan nonsmoker; we found 0.96%. At 50 ppm CO we had 2.96%. At 125 ppm CO we had 6.6%; at 200 ppm CO we had 10.35%; and at 250 ppm CO we had 12.37%. The last two points representing only 5 and 3 subjects might be less accurate, but the spread, for instance with normal nonsmokers at zero conditions, (mean was 0.96%) spread was less than 0.1, the total range. Nice and tight, except for the smoker and he was way off. He had a 3.7%, I believe, at zero exposure.

DR. HODGE: May I ask how long it takes for the carboxyhemoglobin curve to get up to near its 3-hour value?

MAJOR THEODORE: As far as our study was concerned, the 3-hour value that we got was predicted by the 3-hour value plotted by the Forbes-Roughton curves. To reach equilibrium, I can't say for sure. I would guess, and this is only a guess on my part, that it would take at least over 24 hours to reach equilibrium. But, prior to that time, it's rate of uptake of carbon monoxide associated with the affinity with hemoglobin, and you have to take other factors into account--ventilation, cardiac output, probably pH effects in the blood. You probably don't reach steady state carboxyhemoglobin level at exposure to a certain carbon monoxide level in the environment until probably after one day of exposure, but this is only a guess on my part. But before that time, it is really an hourly increment.

DR. MIKULKA: I think it's extremely difficult to estimate with the low levels that we are using, 50 ppm, how long it would take or how high it would go.

MAJOR THEODORE: For any of you who would like to look at the curves, there is one in the Handbook of Physiology and Respiration, Volume II.

DR. BENJAMIN (NASA, Washington): Do you consider the effect of carbon monoxide as a specific toxic effect or is it an indirect anoxic effect?

DR. MIKULKA: The mechanism is supposed to be--tissue hypoxia. That's all I can offer as a psychologist, and from what I read from medical specialists in the area, it should be a hypoxic phenomenon, basically. It may be a lot more than that. We don't show any effect, at this level.

CAPTAIN HICKS (U.S. Army Research Institute on Environmental Medicine): I was curious about your choice of a trial by trial analysis of the data, particularly in the time estimation task. I wondered if you would have a different result if you would analyze all the data together.

DR. MIKULKA: We did. We did everything possible. You see, initially, biased like all experimenters who repeat previous work, we said, "We know there's an effect there", and we analyzed everything. We did what Pierson says is dangerous. We analyzed everything. We found nothing.

DR. PFITZER (University of Cincinnati): As you undoubtedly know, there's a great deal of activity regarding setting environmental quality standards for carbon monoxide, and much of this is based on some of the work with which your data do not agree, and undoubtedly, this will lead to the need for further experiments to see if they duplicate your work. I wonder if you have some thoughts on different kinds of performance tasks which you think might be of more value if further work on this question were to be done.

DR. MIKULKA: I think the problem there becomes what areas are these operational limits being set? The population as a whole? For specific aerospace systems? Cardio-vascular patients? If you go to space systems, you should use tasks like the pilots of a probable spacecraft on a trip to Mars (which is being projected) would be required to be performed, and I can't be specific unless you throw me a task or type of environment. For the psychologist, for the general population, I'd be concerned with tasks involving intellectual functioning, whether these mental functionings are sharp. All data on intellectual functioning, per se, involve crossing T's in a letter, and reduction to absurd limits, and it's a bad area. That's why factor analysis was suggested. A lot of things should be done that haven't been done.

DR. BACK: I think one of the strong points of this particular experiment is the fact that we used a tracking task at which the individual exposed could not win. The faster he tasked himself the more the guts of the system made him fail. The better he got, the worse he could get, so that he was working against a real handicap. He had to fail, and this, I think, gives you some indication of the fine muscular coordination needed, and the intensity at which the man had to work. This is a fine task for a toxicological parameter in evaluating pilot performance.

DR. MIKULKA: This task was tested and confirmed to be a fairly accurate estimation of tracking performance in auto driving, and in piloting high performance aircraft. It's a very involved tracking test. I think it's a good one, not just simply following a little stylus around, but requires rapid reaction to a dial, very quick responses, and visual input with motor output. It's very difficult, I think.

MR. BIBIE (General Electric Company): This series of tests was what I would call a short term series of tests. It was only three hours. How would your test results change if you ran them for a longer time like 24 hours?

DR. MIKULKA: I think the answer to the question is we don't know. For one, we don't know the uptake curves. They haven't been established yet. At three hours, there are no effects. We go as high as 12.37% CO Hb and there are no effects observed. As a psychologist I would say a man could go, 24 hours under 50-125 ppm and show no observable effects, in the types of tasks we have here. Whether you can go for 500 days, back and forth on a spacecraft mission for instance, is another story, and I think it should be tested, at least for some longer terms of exposure.

MAJOR THEODORE: If we did a similar study, as Dr. Mikulka just described, and ran it over 24 hours (and we might) we would be dealing with a different situation because the carboxyhemoglobin levels would be much higher than they were at any given point, but if you go to the higher levels, like he pointed out, 13% carboxyhemoglobin, and were able to go back to the lower levels at equilibrium, I would say maybe you could go at 50 ppm indefinitely if the sole factor involved in decrement were the carboxyhemoglobin level. You have to try to relate it to what you have available, but if you run experiments longer, over 24 hours, it's just really a function of how high the carboxyhemoglobin levels are going to go and they have already reached their limits depending upon the environment, and some of our high points really give credence to our low points as far as exposure is concerned for long periods of time. I would be willing to say that at 50 ppm, from what little I know, some would probably go on indefinitely.



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